CHAPTER 60
Miscellaneous Cranial Nerves

KEY TEACHING POINTS

- Abnormalities of the cranial nerves may reflect local disorders of the nerve itself, injury to its nucleus in the brainstem, or cerebral hemispheric disease interrupting the supranuclear input to the cranial nerve nucleus. Cerebral hemispheric disease must be bilateral before there are motor abnormalities of cranial nerves V, IX, or X.
- Diagnosis of many well-defined syndromes depends entirely on the clinician's observation of cranial nerves: Foster Kennedy syndrome, herpes zoster ophthalmicus, Bell palsy, pseudobulbar palsy, and jugular foramen syndrome are examples.
- In patients with herpes zoster ophthalmicus (i.e., zoster infection of V1, the ophthalmic branch of cranial nerve V), Hutchinson sign (i.e., involvement of the tip of the nose) increases probability of future ocular complications. Even so, all patients with this infection—whether or not Hutchinson sign is present—should receive antiviral medications and be examined by an eye specialist.
- In patients who have had a stroke, several findings increase the patient's risk of aspiration, including the findings of drowsiness, positive water swallow test, and oxygen desaturation of 2% or more after swallowing water. Normal pharyngeal sensation decreases probability of aspiration.

Table 60.1 reviews the physical examination of the 12 cranial nerves. Only cranial nerves I, V, VII, and IX through XII are discussed in this chapter. Cranial nerve II is discussed in Chapters 21 and 58; cranial nerve VIII, in Chapter 24; and cranial nerves III, IV, and VI, in Chapter 59.

OLFACTORY NERVE (I)

I. TECHNIQUE

The usual test for the sense of smell is placing a nonirritative substance, such as wintergreen or cloves, under one nostril at a time. One simple method uses the standard 70% isopropyl alcohol pad available in most clinics and wards.[1] Pungent substances like ammonia should be avoided because they stimulate trigeminal nerve endings (i.e., cranial nerve V).

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TABLE 60.1 The Twelve Cranial Nerves

Cranial Nerve	Motor Examination	Sensory Examination	Reflex Examination
Olfactory nerve (I)		Detection of nonirritating odors	
Optic nerve (II)		Visual acuity Retinal examination	Afferent pupillary defect (swinging flashlight test)
Nerves of the eye muscles: Oculomotor nerve (III) Trochlear nerve (IV) Abducens nerve (VI)	Extraocular movements (III, IV, and VI) Lid elevation (III only)		Pupillary constriction (III only)
Trigeminal nerve (V)	Masseter muscle Lateral pterygoid muscle	Pain, temperature, and touch sensation of the ipsilateral face	Corneal reflex (afferent limb) Jaw jerk (afferent and efferent limb) Glabellar reflex (afferent limb)
Facial nerve (VII)	All facial movements except lid elevation	Taste sensation to anterior two-thirds of the tongue	Corneal reflex (efferent limb) Glabellar reflex (efferent limb)
Vestibulocochlear nerve (VIII)		Tests of hearing (cochlear component)	Vestibuloocular reflex (vestibular component)
Glossopharyngeal nerve (IX)	Ipsilateral palate elevation (with X)	Sensation posterior pharynx Taste sensation to posterior two-thirds of tongue	Gag reflex (afferent limb and, with X, efferent limb)
Vagus nerve (X)	Ipsilateral palate elevation (with IX)		Gag reflex (efferent limb with IX)
Spinal accessory nerve (XI)	Trapezius muscle Sternocleidomastoid muscle		
Hypoglossal nerve (XII)	Genioglossus muscle		

II. CLINICAL SIGNIFICANCE

A. ANOSMIA

Anosmia is the complete absence of smell. The most common causes are upper respiratory infection and sinus disease (which obstructs the nasal passages) and previous head trauma (which damages the olfactory fibers).[2,3] Less common causes are **Kallman syndrome** (hypogonadotropic hypogonadism) and sphenoid ridge masses (e.g., meningioma, which causes the **Foster Kennedy syndrome**, i.e., ipsilateral anosmia, ipsilateral optic atrophy, and contralateral papilledema).[2,4]

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B. OLFACTORY DYSFUNCTION

Patients with olfactory dysfunction are able to detect odors but frequently misidentify them. Olfactory dysfunction is common in patients with Parkinson disease or after frontal or temporal lobectomies.[5,6] Patients with Parkinson disease are much more likely to have olfactory dysfunction than patients with other parkinsonian syndromes, such as vascular parkinsonism and progressive supranuclear palsy (see Chapter 66).[6-10]

TRIGEMINAL NERVE (V)

I. INTRODUCTION

The trigeminal sensory and motor nuclei are located in the pons, although the sensory nucleus extends through the medulla into the cervical spinal cord. The sensory branches of the trigeminal nerve innervate the upper face (V1, ophthalmic division), mid-face (V2, maxillary division), and lower face (V3, mandibular division). The motor fibers to the masseter and lateral pterygoid muscles travel with the mandibular division (V3).

II. THE FINDING

A. MOTOR WEAKNESS

Lesions of the motor component of the trigeminal nerve affect the masseter muscle (causing difficulty clenching that side of the jaw, sometimes with atrophy that flattens the contour of the cheek) and lateral pterygoid muscle (causing difficulty deviating the jaw to the opposite side; at rest, the jaw may deviate toward the weak side).

B. SENSORY LOSS

Lesions of the sensory component cause diminished pain, temperature, and touch sensation in any or all of the three divisions on one side of the face. Sensation to most of the external ear (excluding the tragus) and the angle of the jaw is preserved in trigeminal lesions because these areas are supplied by cervical sensory roots (see Fig. 62.1 in Chapter 62).

C. CORNEAL REFLEX

Unilateral gentle stimulation of the cornea normally causes bilateral blinking. The afferent limb of this reflex is the ipsilateral trigeminal nerve (only V1 and V2) and the efferent limb is both facial nerves (i.e., both eyes blink after stimulation of one cornea).

III. CLINICAL SIGNIFICANCE

A. MOTOR WEAKNESS

Unilateral weakness of the trigeminal muscles indicates disease of the proximal mandibular division (e.g., skull metastases) or a lesion in the ipsilateral pons (patients with pontine lesions have other associated neurologic findings, such as abnormalities of cranial nerves VI or VII, or contralateral hemiparesis). Unilateral weakness

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of the trigeminal muscles does not occur with cerebral hemispheric lesions because each trigeminal nucleus receives bilateral cortical innervation.[11] However, *bilateral* weakness may occur in bilateral cerebral hemispheric disease and cause great difficulty chewing (see the section on Pseudobulbar Palsy).

B. SENSORY LOSS

Sensory loss of the face may be part of a broader neurologic syndrome affecting sensation of the whole body and other neurologic functions (lesions of the cerebral hemisphere, thalamus, or brainstem) or may be isolated to the face (lesions of the peripheral nerve and its branches).

1. SENSORY LOSS OF FACE AND BODY

In thalamic and cerebral hemispheric lesions, sensation of the face and body is abnormal on the *same* side, contralateral to the lesion. There is often associated hemiparesis, aphasia, or both. In brainstem lesions the sensory abnormalities of the face and body are on *opposite* sides: sensation is diminished on the *ipsilateral* face but *contralateral* body (Fig. 62.2 and Table 62.2 in Chapter 62). Pontine lesions affect intraoral more than facial sensation, whereas medullary lesions affect facial more than intraoral sensation.[12]

2. SENSORY LOSS ISOLATED TO THE FACE

Sensory loss isolated to the face is part of syndromes affecting the apex of the temporal bone (see Chapter 59, cranial nerve VI), the cavernous sinus syndrome (V1 division only, see Chapter 59), and **numb chin syndrome**. The numb chin syndrome describes the loss of sensation on the lower lip and chin, an ominous finding in cancer patients because it suggests metastatic disease to the ipsilateral mandible, base of skull, or leptomeninges.[13,14] Some affected patients also have other cranial nerve abnormalities.

C. ABNORMAL CORNEAL REFLEX

The two limbs of the corneal reflex are cranial nerves V and VII. According to traditional teachings, unilateral trigeminal nerve dysfunction (i.e., in the ipsilateral brainstem, V1, or V2 divisions) prevents both eyes from blinking after stimulation of the ipsilateral cornea, whereas unilateral facial nerve dysfunction prevents the ipsilateral eye from blinking when its cornea is stimulated, although the contralateral eye blinks normally. The absent corneal reflex is felt to be particularly important in patients with unilateral sensorineural hearing loss, in whom it raises the possibility of cerebellopontine angle tumors, such as acoustic neuroma.

Nonetheless, the clinical utility of the asymmetric corneal reflex is limited. The reflex is inexplicably absent unilaterally in 8% of healthy elderly patients,[15] and the sensitivity of the absent reflex for acoustic neuroma is only 33%, the finding usually indicating the tumor has already grown to a large size (more than 2 cm in diameter).[16]

D. HERPES ZOSTER INFECTION AND THE NASOCILIARY BRANCH OF THE TRIGEMINAL NERVE ("HUTCHINSON SIGN")

Approximately half of patients with *herpes zoster* infection of the ophthalmic division of the trigeminal nerve (**herpes zoster ophthalmicus**) develop vision-threatening complications, such as uveitis or keratitis within 1 to 4 weeks of the onset of the rash (mean onset of ocular complications is 11 to 13 days).[17-19] In 1865 Hutchinson noted that the tip of the nose, cornea, and iris all share the same branch of the

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trigeminal nerve (the nasociliary nerve) and that if patients with herpes zoster ophthalmicus develop vesicles on the tip of the nose (i.e., **Hutchinson sign**), they were at increased risk of ocular complications.[20] Nonetheless, the clinical utility of this sign is limited: its accuracy is only modest (sensitivity = 43% to 84%, specificity = 76% to 90%, positive likelihood ratio [LR] = 3.3, negative LR = 0.3),[17-19,21,22] and today all patients with herpes zoster ophthalmicus, whether or not the tip of the nose or the eye is involved, should receive antiviral medications and be examined by eye specialists.

FACIAL NERVE (VII)

I. THE FINDING

Lesions of the facial nerve may cause facial asymmetry (diminished ipsilateral nasolabial fold and widened ipsilateral palpebral fissure) and weakness of most ipsilateral facial muscles (muscles used during speaking, blinking, raising eyebrows, smiling, wrinkling the forehead, closing the eyes, showing teeth, and retracting the chin). There may be abnormalities of ipsilateral tearing (lacrimal gland), hearing (stapedius muscle), taste (anterior two-thirds of the tongue), and the corneal and glabellar reflexes.

Facial nerve lesions do not cause ptosis, because the lid muscles are not innervated by the facial nerve but rather by sympathetic nerves and cranial nerve III.

II. CLINICAL SIGNIFICANCE

A. CENTRAL VERSUS PERIPHERAL FACIAL WEAKNESS

Unilateral facial weakness may be *central* (i.e., in upper motor neurons, from lesions in the contralateral motor cortex or descending pyramidal tracts) or *peripheral* (i.e., in lower motor neurons, from lesions in the peripheral nerve or facial nucleus in the ipsilateral pons).* These lesions are distinguished by the following two features:

1. **Distribution of Weakness.** Peripheral lesions affect both upper and lower facial muscles, whereas central lesions affect predominately the lower facial muscles. Wrinkling of the forehead is relatively spared in central lesions because the facial nuclei innervating these muscles receive bilateral cortical innervation.
2. **Movements Affected.** Peripheral lesions paralyze all facial movements on the side affected, whereas central lesions affect voluntary movements but spare emotional ones. The patient with central weakness (e.g., cerebral hemispheric stroke) may be unable to wrinkle one corner of the mouth volitionally yet is able to move it briskly when laughing or crying. This occurs because emotional input to the facial nuclei does not come from the motor cortex.[23,24†]

B. PERIPHERAL NERVE LESIONS

1. ETIOLOGY

The causes of isolated peripheral facial palsies are idiopathic (50% to 87%), surgical or accidental trauma (5% to 22%), herpes zoster infections (**Ramsay Hunt syndrome**, 7% to 13%), tumors (e.g., cholesteatoma, parotid tumors, 1% to 6%), and

*Chapter 61 defines upper and lower motor neurons.

†The opposite clinical finding, emotional paralysis without volitional paralysis, occurs with lesions of the thalamus or frontal lobe.[23,24]

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miscellaneous disorders (8% to 11%). (These figures originate from specialty referral centers and may overrepresent unusual etiologies.)[25-30] **Bell palsy** refers to the idiopathic disorders, although evidence suggests it may represent a viral infection.[31]

2. ASSOCIATED FINDINGS

In patients with Bell palsy, associated findings are diminished taste (52%), hyperacusis (8% to 30%), increased tearing (19% to 34%), and decreased tearing (2% to 17%).[26-29,32-34] Increased tear production occurs because the weak orbicularis oculi muscle cannot contain and direct the tears down the nasolacrimal duct; decreased tearing reflects lacrimal gland dysfunction. Although 23% of patients also have sensory complaints, the finding of hypesthesia of the face (i.e., cranial nerve V) is variable: some investigators, arguing that Bell palsy is part of a multiple cranial neuropathy, have found hypesthesia in as many as 48% of patients,[28,32] whereas other investigators have never found associated hypesthesia of the face.[26]

3. TOPOGRAPHIC DIAGNOSIS

The branches of the facial nerve diverge from the main trunk in predictable order: they are, proximally to distally, branches to the lacrimal gland, stapedius muscle, tongue (taste), and facial muscles.[28] Therefore tests of tearing (Schirmer tear test), stapedius function (stapedius reflex during audiometry), and taste should pinpoint the location of the lesion, although this is only accurate when the nerve is completely severed. In patients with patchy lesions (Bell palsy or partial injuries), topographic diagnosis is often nonsensical (e.g., tearing reduced but taste and stapedius function preserved) and has minimal clinical value.[28,32,35,36]

4. COMPLICATIONS OF BELL PALSY[27,29,33,34]

Three complications occur after recovery from Bell palsy:

1. **Associated Movements.** Associated movements, or **synkinesis**, occur in 55% to 94% of patients. These are unexpected movements that probably result from aberrant regeneration. Examples of associated movements are narrowing of the palpebral fissure when the patient smiles, or motion of the corner of the mouth when the patient tightly closes his or her eyes.
2. **Contracture.** Contracture occurs in 3% to 36% of patients. Despite the name, this actually represents increased muscle tone, not fibrotic scar. Contracture often restores facial symmetry, even though some weakness persists.
3. **Crocodile Tears.** Crocodile tears are seen in 2% to 6% of patients, from aberrant regeneration of salivary gland fibers to the lacrimal gland. When affected patients eat, tears form and run down the cheek or collect in the nose.

GLOSSOPHARYNGEAL (IX) AND VAGUS (X) NERVES

I. FINDING

These nerves are considered together because their function is difficult to separate at the bedside and because clinical disorders usually affect both nerves simultaneously. There are three abnormal findings: (1) **Absent pharyngeal sensation.** This is usually tested with a cotton applicator stick touching the posterior oropharynx; (2) **Diminished velar movement.** The posterior edge of the soft palate is called the **velum** and its elevation, **velar movement**. The soft

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palate should elevate as the patient vocalizes a prolonged "ah"; (3) **Abnormal gag reflex.** During stimulation of the posterior tongue, pharynx, or soft palate, there is reflex elevation of the tongue and soft palate and constriction of the pharyngeal muscles. The gag reflex is labeled abnormal when it is diminished, absent, hyperactive, or asymmetrical.

II. CLINICAL SIGNIFICANCE

Abnormalities of these nerves may occur because of *bilateral* cerebral hemispheric disease or because of disease in the *ipsilateral* medulla or peripheral nerves (i.e., cranial nerves IX and X). *Unilateral* cerebral hemispheric disease does not ordinarily cause palatal weakness because the nuclei of these nerves normally receive bilateral corticobulbar innervation.

A. BILATERAL CEREBRAL HEMISPHERIC LESIONS: PSEUDOBULBAR PALSY

Bilateral lesions above the level of the pons that disrupt the descending pyramidal tracts innervating brainstem motor nuclei may cause significant paralysis of the palate and pharynx, along with paralysis of the tongue, face, and muscles of chewing. This syndrome, **pseudobulbar palsy**, affects approximately 4% of patients with cerebrovascular disease, who mostly have lacunar infarcts in both internal capsules.[37,38] The main clinical features are dysarthria, dysphagia, and paralysis of voluntary movements of the face.[39] Other findings are hyperactive jaw jerk (70% of patients), absent gag reflex (70%), and hyperactive emotional reflexes that cause spasmodic and often inappropriate crying and laughing (24%).[37,38] The animated facial movements during laughter or uncontrollable crying contrast markedly with the lack of voluntary facial movement and the patient's inability to mimic gestures.

The term *pseudobulbar*, coined by Lepine in 1877,[38] is used because the lesion is supranuclear, to distinguish this syndrome from similar motor paralysis that may occur after damage to the brainstem nuclei themselves (i.e., **bulbar paralysis**). The term is a misnomer, however, because *bulbar* refers to the medulla and two of the motor nuclei prominently affected in pseudobulbar palsy—those of the facial muscles (VII) and of chewing (V)—reside in the pons.

B. BEDSIDE PREDICTORS OF RISK OF ASPIRATION AFTER STROKE

In patients who have suffered bilateral strokes, significant dysfunction of cranial nerves IX and X makes the airway vulnerable to aspiration during swallowing. EBM Box 60.1 presents the accuracy of several bedside signs predicting aspiration in patients after strokes. The findings that increase probability of aspiration risk the most are drowsiness (LR = 3.4), abnormal water swallow test (LR = 3.2), and oxygen desaturation of 2% or more after the patient swallows a liquid (LR = 3.1; see footnote to EBM Box 60.1 for definitions of findings). The findings *decreasing* the risk of aspiration the most are normal pharyngeal sensation (LR = 0.03), absence of oxygen desaturation following a swallow (LR = 0.3), and a normal water swallow test (LR = 0.4). The accuracy of other findings, including the abnormal gag reflex, presence of dysphonia, and abnormal cough, is only modest. Findings without predictive value are abnormal sensation of face and tongue, tongue weakness, bilateral cranial nerve findings, and abnormal chest radiograph.[40]

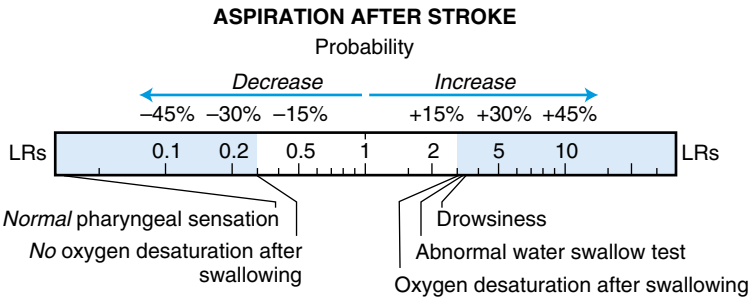
The poor predictive value of the gag reflex is not surprising because the pharyngeal muscles involved in this reflex are not necessarily the same ones activated

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EBM BOX 60.1
*Aspiration After Stroke**

Finding (Reference)†	*Sensitivity (%)*	*Specificity (%)*	Likelihood Ratio‡ if Finding Is	
			Present	*Absent*
Voice and Cough				
Abnormal voluntary cough[40-49]	43-89	36-94	1.9	0.6
Dysphonia[40-45,47,49,50]	54-98	13-85	1.5	0.5
Dysarthria[43,47,51]	60-77	53-57	1.6	0.5
Neurologic Examination				
Drowsiness[48,52]	50-76	65-92	**3.4**	0.5
Abnormal sensation face and tongue[40]	22	52	NS	NS
Absent pharyngeal sensation[52]	98	60	2.4	**0.03**
Tongue weakness[48-50]	50-72	47-91	NS	0.6
Bilateral cranial nerve signs[40,45]	71-73	30-39	NS	NS
Abnormal gag reflex[40-45,47-51]	53-91	18-82	1.4	0.6
Other Tests				
Water swallow test[46-48,50,52,53]	47-85	58-93	**3.2**	0.4
Oxygen desaturation 0-2 min after swallowing[46,54,55]	73-87	39-88	**3.1**	**0.3**

*Diagnostic standard: for *aspiration*, fiberoptic examination[46] or videofluoroscopy (all other studies).
†Definition of findings: for *abnormal voluntary cough,* the patient is asked to cough as hard as possible and the resulting cough is absent, weak, breathy, or sluggish; for *dysphonia*, the patient is asked to sing a prolonged "ah" and the voice is breathy, hoarse, wet, harsh, or strained; for *absent pharyngeal sensation,* the patient cannot sense an applicator stick applied to the posterior oropharynx, on one or both sides; for *abnormal gag reflex,* the gag reflex is diminished, absent, hyperactive, or asymmetric; for *water swallow test*, drinking 5 to 90 mL of water in 5 to 10 mL sips causes coughing, choking, or alteration of the voice; for *oxygen desaturation after swallowing,* oxygen saturation decreases greater than or equal to 2% 0 to 2 min after swallowing 10 mL of water[46] or 20[55] to 150 mL[54] of liquid barium.
‡Likelihood ratio (LR) if finding present = positive LR; LR if finding absent = negative LR.
NS, Not significant.
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during normal swallowing to protect the airway. Moreover, the gag reflex is often absent in normal individuals, especially elderly patients.[56,57] Pharyngeal sensation, on the other hand, is rarely absent in normal individuals.[56]

C. LESIONS OF IPSILATERAL BRAINSTEM OR PERIPHERAL NERVE

The lateral medullary syndrome causes ipsilateral absence of pharyngeal sensation and reduced velar elevation, associated with the Horner syndrome and other sensory and cerebellar signs (see Table 62.2 in Chapter 62). The jugular foramen syndrome (e.g., basilar skull fracture or glomus jugulare tumors) simultaneously disrupts cranial nerves IX, X, and XI, causing ipsilateral paralysis of the palate, vocal cords (hoarseness), and trapezius and sternocleidomastoid muscles.

SPINAL ACCESSORY NERVE (XI)

I. FINDING

The primary findings are a weakness or atrophy, or both, of the sternocleidomastoid muscle (which turns the head to the opposite side) and trapezius muscle (which elevates the ipsilateral shoulder).

II. CLINICAL SIGNIFICANCE

Unilateral weakness of these muscles may represent disease of the cerebral hemispheres, brainstem, spinal cord, or peripheral nerve. Atrophy indicates that the lesion is in the nucleus (i.e., brainstem or high cervical spinal cord) or peripheral nerve (i.e., the lesion is *not* in the cerebral hemispheres).

A. CEREBRAL HEMISPHERE

Lesions of the cerebral hemispheres affect the trapezius and sternocleidomastoid muscles differently: lesions in one cerebral hemisphere weaken the *contralateral* trapezius but the *ipsilateral* sternocleidomastoid muscle.[58‡] Therefore in a hemispheric stroke the patient may demonstrate weakness turning the head toward the hemiparetic side.[59] In a focal seizure the head often deviates toward the seizing limbs.

B. BRAINSTEM OR HIGH CERVICAL SPINAL CORD

Lesions of the accessory nucleus, located in the medulla and high cervical spinal cord (e.g., syringomyelia), may cause atrophy and weakness of the *ipsilateral* trapezius and sternocleidomastoid muscle.

C. PERIPHERAL NERVE

Injuries to the peripheral nerve, which occur from trauma to the posterior triangle of the neck (e.g., surgical excision of lymph nodes, blunt trauma), may paralyze the *ipsilateral* trapezius or sternocleidomastoid muscles, although the sternocleidomastoid

‡ Descending corticobulbar fibers to the sternocleidomastoid muscle are believed to cross twice to innervate the ipsilateral side. This innervation makes teleological sense because the sternocleidmastoid muscle turns the head to the opposite side, and the cerebral hemisphere is interested in turning the head to same side for which it controls visual fields, eye movements, and motor function it controls.[58]

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muscle is often spared because its branches diverge proximally from the main trunk of the nerve.[60] The **jugular foramen syndrome**, discussed above under the glossopharyngeal nerve, also affects cranial nerve XI along with cranial nerves IX and X.

HYPOGLOSSAL NERVE (XII)

I. FINDING

During protrusion of the tongue, each genioglossus muscle acts to push the tongue out and toward the opposite side. Normally these laterally directed forces balance each other, and the tongue remains in the midline. With unilateral hypoglossal weakness, however, the intact genioglossus muscle acts to deviate the tongue toward the opposite, or weak, side.

II. CLINICAL SIGNIFICANCE

Weakness of the genioglossus may represent disease in the cerebral hemisphere, brainstem, or peripheral nerve. Atrophy or fasciculations of the tongue indicate the lesion is either in the hypoglossal nucleus (brainstem) or hypoglossal nerve (i.e., *not* cerebral hemispheres).

A. CEREBRAL HEMISPHERE

Lesions of the cerebral hemisphere may cause weakness of the contralateral genioglossus. Therefore the tongue deviates *toward* the side of the weak arm and leg.[61]

B. BRAINSTEM

The medial medullary syndrome causes ipsilateral hypoglossal paralysis, contralateral hemiparesis, and contralateral loss of proprioceptive and vibratory sensation (preserving pain and temperature sensation). Therefore the tongue deviates *away* from the side of the weak arm and leg.

C. PERIPHERAL NERVE

The most common causes of lesions of the hypoglossal nerve are metastatic cancer (to base of skull, subarachnoid space, or neck) and trauma (e.g., gunshot wounds to the neck, radical neck surgery, carotid endarterectomy).[62]

Hypoglossal palsy in association with other cranial nerve findings occurs with both brainstem and peripheral nerve disorders and therefore has little localizing value.[62]

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